Beyond Extinction:

Habituation Eliminates Conditioned Skin Conductance Across Contexts

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Abstract

A marked signature of fear extinction is its vulnerability for relapse. Here, we departed from the standard extinction principle and examined the ability of habituation to reduce conditioned fear reactions and prevent relapse. In a human fear conditioning paradigm, we first established one visual stimulus as a signal for an impending aversive electrical stimulation, while another visual stimulus was never followed by this stimulation. Next, the screen color changed and participants were either exposed to the visual stimuli without electrical stimulation (extinction treatment) or to the electrical stimulation without the visual stimuli (habituation treatment). Finally, the screen color changed back and the two visual stimuli were tested. Verbal ratings showed a return of conditioned shock-expectancy in the two groups, while skin conductance reactivity showed conditioned discrimination following exposures to the visual stimuli, but not following exposures to the electrical stimulation. We conclude that an habituation treatment outperforms an extinction treatment, and that shock-expectancy and skin conductance can dissociate under some conditions.

Keywords: fear conditioning, extinction, devaluation, habituation, renewal, skin conductance response

1. Introduction

Fear is an adaptive emotion that motivates the defensive reaction system in the face of danger. An optimal strategy requires the identification of valid signals of danger, which can then trigger fear and motivate preemptive defensive reactions. This is generally referred to as *fear learning* and is modelled by Pavlovian fear conditioning. In this procedure, a neutral stimulus (conditional stimulus, CS) is repeatedly followed by an aversive stimulus (unconditional stimulus, US) and results in de novo fear reactions to the CS. Arguably, as the contingency between these two events is learned, new encounters with the CS come to activate a memory representation of the US. This causes the CS to elicit a conditioned fear response (CR) with an intensity adapted to the aversiveness of this US representation (Davey, 1988). In cognitive terms, then, fear may reflect an interaction between the estimated probability and the estimated intensity of an aversive event:

$$\text{Fear} = \text{Probability} \times \text{Intensity} \qquad (1)$$

In conditioning terms, the estimated probability relates to the construct 'CS-US association' and the estimated intensity to the 'US memory'.

This analysis suggests that exacerbated levels of fear, as in anxiety disorders, are due to an overestimation of probability (CS-US) and/or intensity (US). Most anxiety treatments are explicitly aimed at decreasing exacerbated levels of fear (e.g., Fao & Kozak, 1986; but see Hayes et al., 2006). The most intensively studied technique in this regard is extinction, which refers to the fear reduction observed when the CS is repeatedly presented in the absence of the aversive US. The goal is to weaken/inhibit the CS-US association and hence the estimated US probability. Exposure-based treatments apply this extinction principle by exposing the anxious client to his/her feared situation in the absence of the anticipated aversive outcome

(Myers, & Davis, 2007). These treatments are generally very effective in reducing fear levels in the short term (Butler et al., 1984; Rothbaum et al., 2000, Öst et al., 1993; Vlaeyen et al., 2002), but they suffer from a continuous risk of relapse (return of fear; Vervliet et al., 2013b). Increasing the long-term effectiveness of fear extinction provides the strongest challenge for clinical and pre-clinical research on anxiety. Importantly, Pavlovian fear conditioning studies have revealed that fear extinction is highly context-dependent, and that changes in the surrounding context elicit a return of fear after extinction (e.g., Vansteenwegen et al., 2005). Likewise, changes in context elicit a return of fear following successful exposure treatments (e.g., Rodriguez et al., 1999). Enhancing the generalizability of fear extinction over contexts is therefore a major challenge towards the improvement of the long-term effects of exposure-based treatments. The current study tested a novel technique aimed towards this goal.

Fear extinction research and anxiety treatments focus on weakening the CS-US association (the estimated US probability), but largely neglect the US memory itself. Nevertheless, some studies show that treatments that devalue US memories directly also reduce CS-elicited fear in animals (Storsve, McNally, & Richardson, 2010, 2012) and in humans (Hosoba et al., 2001; Dibbets et al., 2011). Devaluation techniques included (1) repeated exposures to the US (habituation), (2) exposures to reduced levels of the US (deflation), and (3) imagery rescripting (reappraisal). Despite successful fear reduction, however, the effects on contextual renewal are mixed. A series of US-habituation experiments in *rodents* revealed no prevention of renewal, that is, an intact return of CS-fear following a context change (Storsve et al., 2010, 2012). In contrast, combined CS-alone extinction trials with imagery rescripting did reduce renewal of fear in *humans* (Dibbets et al., 2011). Also, combined CS-alone extinction trials with US-alone habituation trials eliminated renewal of fear in humans (Vervliet et al., 2010). Together, these studies leave open the possibility that (1) targeting the US memory is more effective in humans than in rats, or that (2) mixing CS-

extinction with US memory interventions is more effective than either alone. In order to solve this dual possibility, the current study was set up to investigate the sole effect of US-habituation on fear renewal in humans (analogous to the studies in rats by Storsve et al. 2010, 2012). We compared this to fear renewal after traditional extinction. Analogous to Vervliet et al., (2010), this study used a contextual renewal procedure to examine return of fear in humans. Following differential fear conditioning with two neutral CSs in context A, half of the participants received CS-alone exposure and half received US-alone exposure in context B. Finally, both CSs were presented again in context A in order to measure the amount of return of fear. The only difference with Vervliet et al. (2010) was the removal of CS-alone trials in the CS/US unpaired group of that study.

Of interest, we measured both US-expectancy ratings and skin conductance reactivity during CS presentations. We hypothesized that US-expectancy ratings are valence-free and can track the strength of the estimated US probability (CS-US association) irrespective of the estimated US intensity (US memory). Skin conductance reactivity, on the other hand, depends on both the estimated probability and intensity of the US. Therefore, we expected strongly renewed expectancy of the US in both groups, and a return of conditioned skin conductance only in the CS-exposure group.

2. Material and methods

2.1 Participants

First-year psychology students and community volunteers participated in return for payment (8 euro) or course credits. Data from two independent but identical replications of the same experiment were merged. This resulted in a total sample of eighty-seven participants (sixty-two women) with a mean age of 20.9 (*SD* = 4.70). Participants were randomly assigned

to one of two groups. All participants gave informed consent and were aware that they could abort the experiment at any time.

Apparatus

2.2 Conditioned stimuli and contexts

Two geometrical shapes (square and triangle) served as conditional stimuli (CS1 and CS2) and were presented on a computer screen (Dell LCD monitor, type 1707 FPc). These shapes were grey with a black border and presented in a white frame. Stimuli slightly differed between the two experiments. In the first experiment, stimuli were darker grey and the white frame was square (versus rectangular in the second experiment). The background context was manipulated by altering the color of the background of the computer screen between yellow (RGB 255, 255, 128) and blue (RGB 0, 255, 255).

2.3 Unconditioned stimulus

The US was a 2 ms electrocutaneous stimulus administered to the wrist of the dominant hand. It was administered by a Digitimer DS7A constant current stimulator (Hertfodshire, UK) via a pair of V91-01-8mm reusable Bilaney Ag/AgCL electrodes. These electrodes were filled with K-Y Jelly.

2.4 Skin conductance reactivity

Electrodermal activity was recorded using a skin conductance coupler manufactured by Coulbourn Instruments (model V71-23, Allentown, PA). The coupler applied a constant voltage of 0.5 V across a pair of 8mm Ag/AgCl electrodes. These electrodes were attached to the palm of the non-dominant hand. The resulting skin conductance signal passed through a Labmaster DMA 12 bit analog-to-digital converter (Scientific Solutions, Solon, Ohio) and digitized at 10 Hz from 2 s prior to CS onset until 6 s after CS offset.

2.5 US-expectancy

An eleven-point scale was used to measure trial-by-trial subjective shock expectancy ratings. The scale ranged from 0 to 10 and was labelled: “certainly no shock” (0), “maybe” (5), “certain shock” (10). A left mouse click on the scale registered the corresponding position for that trial.

The stimulus sequence, stimulus presentation, ITI, and response registration was controlled by Affect 4.0 software (Hermans et al., 2002).

2.6 Procedure

After participants gave their informed consent electrodes were fitted and the shock intensity was set to a level that was determined “definitely uncomfortable, but not painful” through a standard shock work-up procedure. Subsequently, participants were instructed that pictures of geometrical shapes would appear on the computer screen and that some of these shapes could be followed by a shock. It was further explained that the participant’s task was to predict the occurrence of the shock. Next, participants were instructed how to use the expectancy ratings scale.

The experiment consisted of four phases (see Table 1). The experiment started with a non-reinforced presentation of CS1 and CS2 in order to weaken the initial orienting responses to these stimuli (pre-acquisition). During acquisition, each stimulus was presented four times in context A. CS1 was always followed by shock, CS2 never. The geometrical shapes serving as CS1 and CS2 were counterbalanced. Following acquisition, the screen color changed (context B) and participants in the CS-exposure group received traditional extinction training (eight presentations of CS1 and CS2 without reinforcement). Participants in the US-exposure group received eight presentations of the shock. Time between two shock administrations differed slightly between the two experiments, 23.43 seconds (range 22-26 s) in Experiment 1 and 22 seconds (range 20-24 s) in Experiment 2. Finally, the screen changed back to its original color (context A) and each CS was presented three times without shock. The order of

context was counterbalanced; for half of the participants the order was yellow – blue – yellow, versus blue – yellow – blue for the other half.

Throughout the experiment, CS duration was always eight seconds; with on average 14 s (range 12-16 s) intertrial interval (from CS offset to CS onset). The scale appeared at the bottom of the screen at CS onset. Participants used the computer mouse to control a red dot on the scale and indicate their rating. Once participants gave a rating, the scale disappeared from the screen.

2.7 Data reduction

Due to recording error, expectancy ratings and SCR from one participant were excluded from data analysis. A second participant failed to respond within the given time frame (8 s) resulting in no registered expectancy ratings. Visual inspection of the skin conductance responses also revealed recording errors with four additional participants, who were subsequently excluded from data analyses as well. Finally, expectancy ratings and SCR from the pre-acquisition familiarization trials were not included in the analyses.

Skin conductance responses were extracted with the PSPHA software for analyzing psychophysiological data (De Clerck, Verschuere, De Vlieger, & Crombez, 2006). Skin conductance response magnitudes were calculated and standardized according to guidelines in Dawson et al. (2007). SCR magnitudes to the CS were calculated by determining the maximal increase in skin conductance between 900 ms and 7000 ms following CS onset. For the US habituation phase, skin conductance responses magnitudes for the US were calculated by determining the largest increase in skin conductance between 900 ms and 6500 ms (the end of the preset measurement window) following shock administration. Next, to reduce interindividual variability, all values for the acquisition and test phase were T-transformed

over all acquisition and test trials for each participant[1]. CS magnitudes for the extinction phase were T-transformed over all extinction trials for each participant in the CS-exposure group). For the US-exposure group, US magnitudes were T-transformed over all habituation trials for each participant.

3. Results

3.1 Shock expectancy ratings

3.1.1 Acquisition

The left panel of Figure 1 suggests a gradual increase in shock-expectancy for CS1 and a gradual decrease for CS2 over the acquisition trials in both groups. Accordingly, a 2 (Group) x 2 (CS) x 4 (Trial) RM-ANOVA revealed a significant main effect of CS, $F(1, 78) = 375.29$, $p < .001$, $\eta^2_{part} = .82$, and a significant CS x Trial interaction, $F(2.21, 172.18) = 236.69$, $p < .001$, $\eta^2_{part} = .75$, with significant linear, $F(1, 78) = 551.11$, $p < .001$, $\eta^2_{part} = .88$, and quadratic component, $F(1, 78) = 51.90$, $p < .001$, $\eta^2_{part} = .40$. Importantly, there was no main effect of Group, $F(1, 78) = 0.58$, $p = .45$, nor any interaction with Group (Fs < 0.97, ps $> .447$). This suggests similarly successful acquisition rates in both groups.

3.1.2 Generalization of acquisition (Group CS-exposure)

The middle panel of Figure 1 suggests that shock-expectancy remained high during CS1 in a novel context, while responding to the CS2 increased considerably. A 2 (CS) x 2 (Trial) RM-ANOVA indeed revealed a significant CS x Trial interaction, $F(1, 40) = 81.98$, $p < .001$, $\eta^2_{part} = .67$. Post-hoc comparisons revealed that shock-expectancy for the CS1 significantly decreased from the last training trial to the first extinction trial, $F(1, 40) = 26.62$, $p < .001$, $\eta^2_{part} = .40$, and significantly increased for the CS2, $F(1, 40) = 81.18$, $p < .001$, η^2_{part}

[1] SCR amplitudes for the acquisition and test phase were T-transformed over acquisition and test trials only (and not extinction trials for group extinction), in order to preserve comparability between both groups.

= .67. Importantly, stimulus discrimination remained significant on the first extinction trial in the novel context, $F(1, 40) = 17.36$, $p < .001$, $\eta^2_{part} = .30$. This indicates a certain degree of generalization of the conditioned differential shock-expectancy to the new context.

3.1.3 Extinction (Group CS-exposure)

The middle panel of Figure 1 suggests a gradual decrease in the conditioned discrimination. This was confirmed by a 2 (CS) x 8 (Trial) RM-ANOVA, revealing a significant main effect of CS, $F(1, 40) = 17.00$, $p < .001$, $\eta^2_{part} = .30$ and Trial, $F(3.004, 120.150) = 98.03$, $p < .001$, $\eta^2_{part} = .71$. The CS x Trial interaction was also significant, $F(3.97, 158.93) = 7.48$, $p < .001$, $\eta^2_{part} = .16$, with a significant linear, $F(1, 40) = 22.19$, $p < .001$, $\eta^2_{part} = .36$, and quadratic trend, $F(1, 40) = 7.21$, $p = .01$, $\eta^2_{part} = .15$.

3.1.4 Test

The right panel of Figure 1 suggests differential shock-expectancy during CS1 versus CS2 on the three test trials, for both groups. This was confirmed by a 2 (CS) x 3 (Trial) x 2 (Group) RM-ANOVA, which revealed a main effect of CS, $F(1, 81) = 80.29$, $p < .001$, $\eta^2_{part} = .50$. Overall, shock-expectancy ratings were higher in group US-exposure, main effect of Group, $F(1, 81) = 14.56$, $p < .001$, $\eta^2_{part} = .15$. Moreover, the CS1/CS2 discrimination was larger in Group US-exposure: CS x Group interaction, $F(1, 81) = 4.11$, $p < .05$, $\eta^2_{part} = .05$. Post-hoc comparisons revealed higher shock expectancies for the CS1 in group US-exposure compared to group CS-exposure, $F(1, 81) = 14.49$, $p < .001$, $\eta^2_{part} = .15$, and no differences in shock expectancy for the CS2 between groups, $F(1, 81) = 1.44$, $p = .23$. Figure 1 further suggests that after CS-exposure (CS-exposure group), conditioned responding returned when the CS1 and CS2 were presented again in the original acquisition context (contextual renewal). A 2 (CS) x 2 (Trial) RM-ANOVA compared outcome expectancy ratings to CS1 and CS2 on the last extinction trial to ratings on the first test trials. This indeed revealed a significant CS-type x Trial interaction, $F(1, 40) = .20.78$, $p < .001$, $\eta^2_{part} = .34$. Post hoc

comparisons revealed that both the CS1, $F(1, 40) = 81.48$, $p < .001$, $\eta^2_{part} = .67$, and the CS2, $F(1, 40) = 40.87$, $p < .001$, $\eta^2_{part} = .51$, significantly increased from the last extinction trial to the first test trial.

3.2 Skin Conductance Response

3.2.1 Acquisition

The left panel of Figure 2 shows a gradual increase in differential SCR to the CSs for both groups (see supplemental Figure 1 for the raw skin conductance data in μSiemens). This was confirmed by a 2 (CS) x 4 (Trial) x 2 (Group) RM-ANOVA, which revealed a main effect of CS, $F(1, 82) = 8.944$, $p = .004$, $\eta^2_{part} = .10$, and a CS-type x Trial interaction, . $F(3, 246) = 3.79$, $p = .01$, $\eta^2_{part} = .04$, with a significant linear trend, $F(1, 82) = 8.22$, $p = .01$, $\eta^2_{part} = .09$. None of the interactions with Group reached significance (Fs <0.57, ps $> .64$). This suggests similarly successful acquisition rates in both groups.

3.2.2 Extinction (Group CS-exposure)

The middle panel of Figure 2 shows an inconsistent pattern of SCR to both stimuli during extinction in the CS-exposure group, rather than the expected gradual decrease (see supplemental figure 1 for the raw skin conductance data in μSiemens). This was confirmed by a 2 (CS) x 8 (Trial) RM-ANOVA, which revealed no main effect of CS, $F(1, 40) = 1.15$, $p = .29$, no main effect of Trial $F(7, 280) = 1.42$, $p = .20$ and no interaction effect of CS x Trial, $F(7, 280) = 1.36$, $p = .22$.

3.2.3 US-exposure presentations (Group US-exposure)

The middle panel of Figure 2 shows a gradual decrease in SCR to US in group US-exposure (see supplemental figure 1 for the raw skin conductance data in μSiemens). This was confirmed by a RM-ANOVA with one within subjects factor (Trial, 8 levels), which revealed a main effect of Trial, $F(7, 280) = 5.117$, $p < .001$., $\eta^2_{part} = .11$., with a significant linear trend, $F(1, 40) = 24.69$, $p < .001$, $\eta^2_{part} = .38$.

3.2.4 Test

The right panel of Figure 2 shows the conditioned differential response to CS1 and CS2 on the first test trial in group CS-exposure, but not in group US-exposure. Since this effect quickly diminished over the nonreinforced test trials, we restricted our analysis to the first test trial (see supplemental figure 1 for the raw skin conductance data in μSiemens). A 2 (CS) x 2 (Group) RM ANOVA showed no main effect of CS, $F(1, 82) = .69$, $p = .41$, but a significant interaction between CS and Group, $F(1, 82) = 3.98$, $p < .05$, $\eta^2_{part} = .05$. In addition, post hoc comparisons revealed that the CS1 versus CS2 discrimination was significant at trend level in group CS-exposure, $F(1, 82) = 3.90$, $p = .05$, $\eta^2_{part} = .05$, but not in group US-exposure, $F(1, 82) = .69$, $p = .41$.

An additional 2 (Trial) x 2 (Group) RM ANOVA compared responding to CS1 on the last acquisition trial and the first test trial in both groups. This analysis revealed a significant Trial x Group interaction, $F(1, 82) = 4.59$, $p = .03$, $\eta^2_{part} = .05$. Post hoc comparisons showed a boarder significant decrease in SCR to CS1 from the last acquisition trial to the first test trial for group US-exposure, $F(1, 82) = 3.67$, $p = .06$, $\eta^2_{part} = .04$ and no decrease for group CS-exposure, $F(1, 82) = 1.26$, $p = .26$, $\eta^2_{part} = .02$. These results suggest an intact conditioned skin conductance response to CS1 in group CS-exposure, while the response decreased in group US-exposure.

4. Discussion

The present experiment was set up to directly compare the effects of US-exposure versus traditional CS-exposure on the level of fear in a change of context paradigm. During initial fear acquisition, the two groups learned equally well to discriminate between the CS1+ and the CS2-, both in conditioned shock-expectancy ratings and in skin conductance reactivity. Following exposures to the CSs versus the USs in another context, both groups

were tested with CS1 and CS2 in the original acquisition context again. Here, the two measures dissociated. The shock-expectancy ratings showed the conditioned discrimination in both groups, with a significant larger discrimination in the US-exposure group. In contrast, the skin conductance showed the conditioned discrimination in the CS-exposure group, but not in the US-exposure group. For skin conductance, therefore, exposure to the US in a different context effectively eliminated the conditioned discrimination within the conditioning context, while exposures to the CSs did not.

The results in the CS-exposure group relate to contextual renewal, a finding in many preparations and species that fear extinction via CS exposures does not generalize across contexts (Vervliet et al., 2013b). Surprisingly, however, we found no clear evidence for extinction of the skin conductance in the CS-exposure phase, as the conditioned discrimination disappeared immediately on the first extinction trial in the novel context (see also Vervliet et al., 2010, Vervliet et al., 2013a). In principle, this absence of clear extinction may have biased our test comparison in favor of the US-exposure group when it comes to eliminating the conditioned discrimination. Notwithstanding, the wealth of renewal reports in the literature, including conditioned skin conductance in humans, strongly suggests that a clearer extinction effect in our experiment would have had little influence on the degree of return at test (Vervliet et al., 2013b).

The current results also complement an earlier study by Vervliet et al. (2010), who found that combining CS-exposure and US-exposure eliminated renewal of the conditioned discrimination in *both* skin conductance reactivity and shock-expectancy ratings. Together, the two studies suggest that (1) traditional CS exposure leads to renewal of shock-expectancy and skin conductance, (2) US-exposure (habituation) leads to renewal of shock-expectancy, but eliminates renewal of skin conductance, and (3) combined CS-exposure and US-exposure eliminates renewal in both measures. The following theoretical pattern emerges, based on the

Fear = Probability x Intensity formula. Shock-expectancy ratings track the estimated probability (CS-US association), while skin conductance reactivity tracks the interaction between estimated probability and intensity (US memory). First, the CS-exposure treatment (extinction) fails to impact the estimated probability or the estimated intensity (when tested in the acquisition context). Second, US-exposure (habituation) impacts the estimated intensity, but not the estimated probability (because the shock-expectancy ratings are intact). Third, the CS/US unpaired treatment (Vervliet et al., 2010) impacts both the estimated probability and the estimated intensity, in the original acquisition context.

This analysis suggests that CS-exposure and US-exposure act via different mechanisms. This goes against some associative learning theories that posit similar mechanisms underlying the effects of both treatments. First, the fear reduction during CS-exposure might involve a US-deflation mechanism (repeatedly thinking of the US without experiencing it would weaken the US memory; Rescorla & Heth, 1975). Our skin conductance results are at odds with this hypothesis, as the pattern of results from a direct US-habituation procedure is very different. Second, US-exposure might involve a CS-extinction mechanism as the experiences of the US in the absence of the CS reduce their contingency and may therefore weaken the association and estimated probability (e.g., Rescorla & Wagner, 1972). Intact shock-expectancy ratings in the US-exposure group suggest that this is not the case. However, it is still possible that shock-expectancy ratings did reduce in the US-exposure context but renewed in the acquisition context. This awaits further investigation. For now, it seems that combining CS-exposure and US-exposure (1) capitalizes on a US-habituation mechanism that weakens the US memory and estimated intensity, and (2) strengthens the CS-extinction mechanism that weakens/inhibits the CS-US association and estimated probability. This leads to elimination of renewal of both shock-expectancy and skin conductance.

Storvse et al. (2010, 2012) found different habituation results in a fear conditioning procedure in rats. They did observe a reduction of conditioned fear (percentage time spent freezing during CS presentation), but only when the CS was tested in the US-exposure context. Testing the CS in the acquisition context led to a renewal of conditioned freezing, just like an extinction treatment does. This is opposite to our skin conductance results. There are many differences between the procedures that can contribute to this variance. First, there may obviously be a difference in species. Maybe the cognitive framework that we developed here is used by humans but not by non-human animals. Second, Storsve et al. (2010, 2012) tested long-term renewal effects (i.e., 24 hours between treatment and renewal test), whereas we tested short-term renewal effects (no gap between treatment and test). Time has an important influence on memory processes, and may therefore impact the results of US-exposure as well. Third, freezing and skin conductance reactivity may measure different components of the conditioned fear reaction so that they react differently to the treatments. Unfortunately, there is hardly any measure of freezing available for humans (but see Hagenaars, 2010). Fourth, the intensity of the (experience of) electrical stimulation (US) is probably different in the two procedures. It is plausible that the short-term as well as the long-term effects of US-exposure are influenced by the intensity factor.

There is an ongoing debate in the human fear conditioning literature about the validity of different psychophysiological measures of the fear construct. In particular, some researchers question the relevance of skin conductance for the measurement of fear, often proposing the fear-potentiated startle reflex (FPS) as a more accurate index of current fear. For example, Soeter and Kindt (2010, p. 30) state that "… skin conductance conditioning primarily reflects contingency learning, whereas the startle response is a rather specific measure of fear". They also reported a strong association between declarative knowledge (measured as shock-expectancy ratings) and skin conductance, while FPS was dissociated

under some conditions. The current results show in a simple way that skin conductance is not merely a physiological reflection of declarative contingency. US-exposure dissociated the skin conductance from the shock-expectancy ratings. This shows that skin conductance tracks learning about upcoming *threat*. The question then becomes to what extent threat anticipation is a core component of the construct 'fear'. In this sense, the construct 'fear' is highly elusive and may even cause general ambiguity in the field. It may be better to use the term 'threat learning' as this is closer to the procedure that we use and less influenced by conceptual issues (LeDoux, 2014).

Exposure treatments for anxiety disorders are typically viewed as CS-exposure (extinction), but may also involve US-exposure. One challenge for scientist-practitioners is that the CS and the US are often not so clearly identifiable in clinical cases. Take the example of a person who is afraid of elevators following a panic attack inside an elevator. We could term the elevator the CS and the panic attack the US, resulting in fear of elevators. CS-exposure would expose to elevators, while US-exposure would expose to panic attacks (or close proxies, like hyperventilation). Both types of interventions are part of current treatment protocols for panic disorder (e.g., Barlow et al., 1989; Fava et al., 2001). However, a panic attack itself is often not the final US, but is itself associated with other catastrophic outcomes ("I will go crazy", "what would people around me think of me", "I will die of a heart attack"). In that case, the panic attack is a CS that is associated with these other USs. Some of these USs allow for direct exposure, others do not. Also, some anxiety disorders involve 'fear of fear' in the sense that the fear reactions themselves are aversive and fear eliciting (acting as US; Chambless & Gracely, 1989). In these cases, exposure therapy that gradually elicits fear reactions can be considered a US-exposure treatment. This example shows that the CS/US dichotomy starts to blur once we leave the laboratory, and that it becomes less clear which principles underlie the fear reduction effects of exposure treatments. In our view, the current

study highlights the importance of looking both at CS-exposure and US-exposure when modeling exposure treatments in the laboratory.

There are several limitations to this study. First, we had to merge two identical replications of the same experiment in order to obtain enough power. Second, as mentioned earlier, the extinction phase in the CS-exposure group was atypical. The context-change after acquisition (novel screen color) dampened the conditioned discrimination in the skin conductance (but much less so in the shock-expectancy ratings). Consequently, the extinction phase did not show a typical extinction curve with a gradually decreasing discrimination of the skin conductance reaction. Again, the ratings did show the expected extinction curve. Vervliet et al. (2010) reported a similar pattern of results with a highly similar procedure. These atypical extinction findings limit the interpretation of the return of fear results as a standard renewal effect (Vervliet et al., 2013a). Arguably, the color change may have been a salient event that triggered orienting reactions in the skin conductance (Öhman, 1983). Also, we did not use a partial reinforcement procedure, which may prolong conditioned responding during extinction and thereby provide more power to detect an extinction curve.

Third, we did not include post-experimental questions about the US aversiveness (e.g., positive-negative valence ratings). It is unclear, therefore, what the direct effects of US-exposure were on the perceived/remembered intensity of the US.

Finally, the beneficial effect of US-exposure over CS-exposure on renewal of conditioned skin conductance responses was only observed on the first test trial. Replication of these results is therefore needed in order to substantiate the conclusions drawn from the present study.

5. Conclusion

In sum, the current study found that US-exposure can eliminate renewal of conditioned skin conductance reactions, but leaves conditioned shock-expectancy intact. This

shows that a US-exposure has advantages over a CS-exposure and corroborates an earlier study that found a general elimination of renewal after a combination of CS-exposure and US-exposure (Vervliet et al., 2010). Because habituation processes may underlie the current findings, this study calls for more research on habituation as a means to reduce conditioned fear reactions. Also, the results show a marked dissociation between conditioned skin conductance reactivity and conditioned shock-expectancy, thereby challenging conceptualizations of the skin conductance as a mere reflection of declarative memory and/or contingency learning.

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Table 1
Overview of the experimental phases and groups.

	Pre exposure	Acquisition	Fear reduction	Test
Group CS-only	CS1- (1) CS2- (1)	CS1+(4) CS2- (4)	CS1- (8) CS2- (8)	CS1- (3) CS2- (3)
Group US-only	CS1- (1) CS2- (1)	CS1+(4) CS2- (4)	US (8)	CS1- (3) CS2- (3)

Note: CS1 and CS2 are geometrical shapes (counterbalanced); '+' refers to the administration of the shock US; '-' refers to the absence of shock. The number of trial presentations is indicated between parentheses. The background coloring refers to the experimental context (blue or yellow background of the computer screen, counterbalanced)

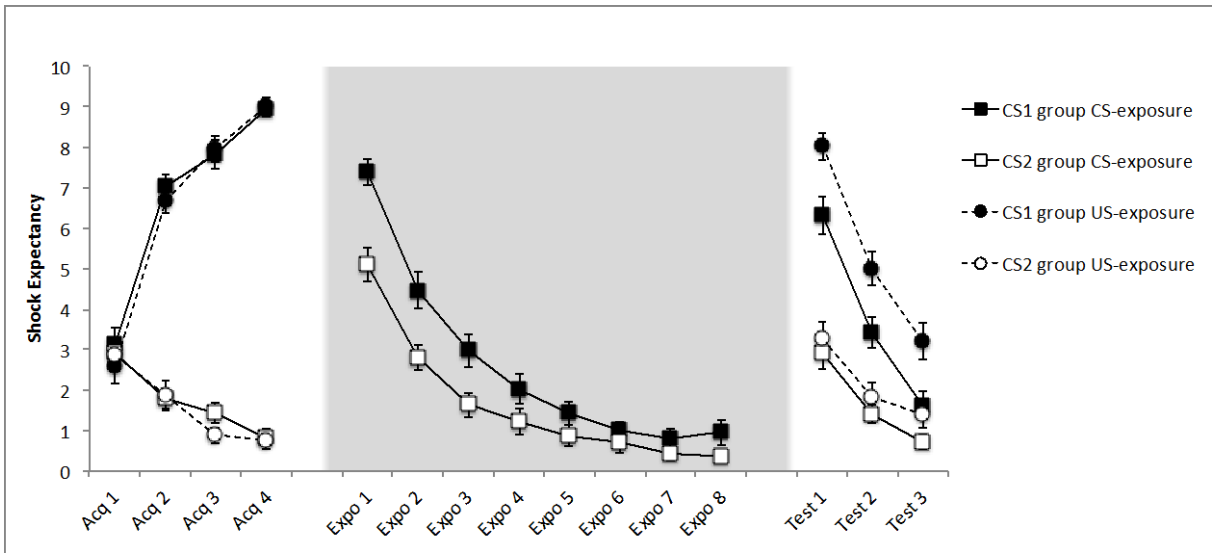


Fig. 1. Mean online shock-expectancy ratings for groups CS-exposure and US-exposure, as a function of CS-type (CS1/CS2) and Trial. Both groups received one CS-preexposure trial (not shown) and four acquisition trials. Three test trials followed eight CS-alone presentations in group CS-exposure and eight US-alone presentations in group US-exposure. No expectancy ratings were registered during the US-alone presentations in group US-exposure. Background colors represent experimental contexts. Error bars represent standard errors of the mean (SEM).

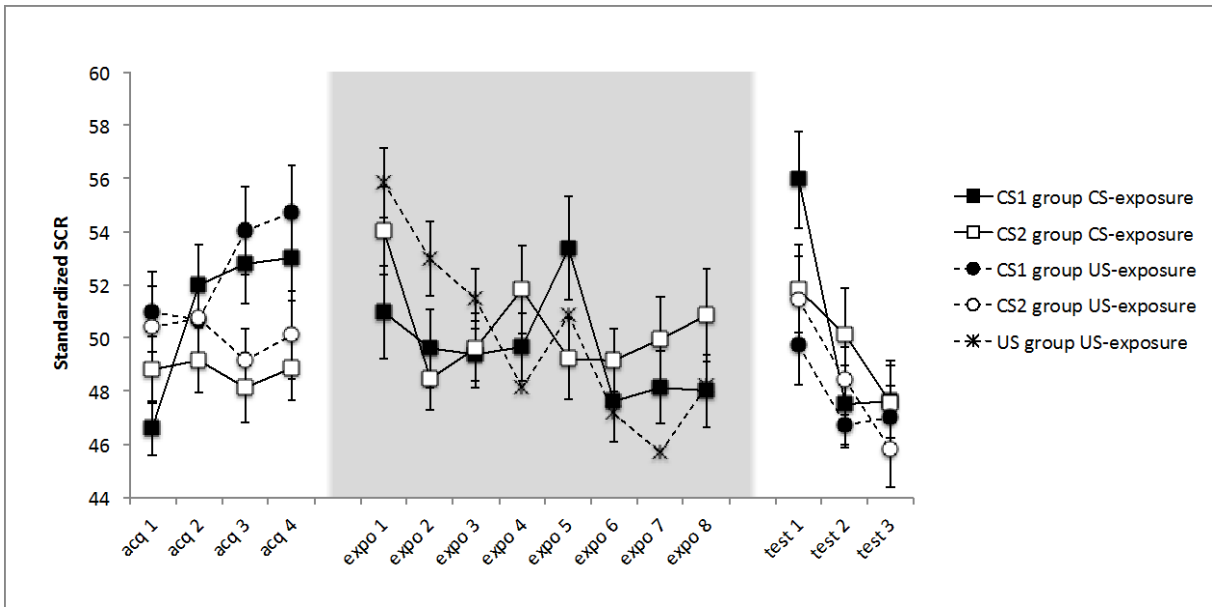
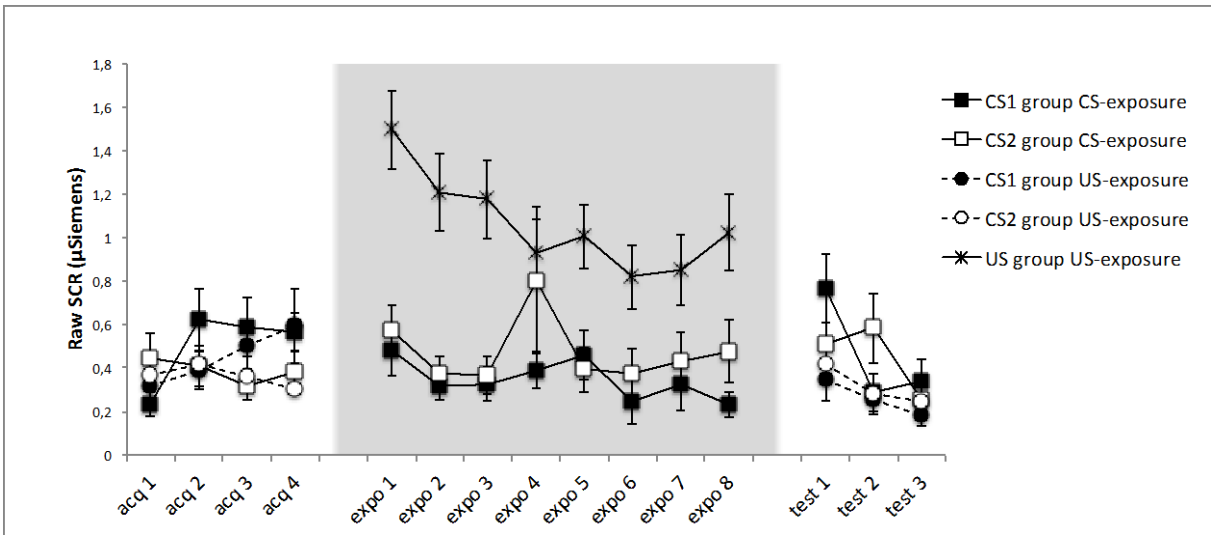


Fig. 2. Mean standardized skin conductance responses for groups CS-exposure and US-exposure as a function of stimulus type (CS1/CS2/US) and Trial. Both groups received one CS-preexposure trial (not shown) and four acquisition trials. Three test trials followed eight CS-alone presentations in group CS-exposure and eight US-alone presentations in group US-exposure. Note that SCR magnitudes were standardized across the acquisition and test phase in both groups. For the intermediate CS-exposure or US-exposure phase, SCR magnitudes were separately standardized over the CS-exposure or US-exposure trials, respectively. Note that standardized results provide no information with regard to absolute differences between groups or between separately standardized phases. Background colors represent the experimental contexts. Error bars represent standard errors of the mean (SEM).



Supplemental fig. 1. Mean raw skin conductance responses (expressed in µSiemens) for groups CS-exposure and US-exposure as a function of stimulus type (CS1/CS2/US) and Trial. Both groups received one CS-preexposure trial (not shown) and four acquisition trials. Three test trials followed eight CS-alone presentations in group CS-exposure and eight US-alone presentations in group US-exposure. Background colors represent the experimental contexts. Error bars represent standard errors of the mean (SEM).